Amendments to the Claims:

This listing of claims will replace all prior versions, and listings of claims in the application:

Listing of Claims:

Claims 1-68 (Canceled).

69. (Previously presented) A chimeric peptide comprising:

a peptide having a first portion and a second portion, wherein the carboxyl terminus of the first portion is linked to the amino terminus of the second portion; and,

wherein the first portion is from the free N-terminus of a naturally-occurring internal peptide cleavage product which, when naturally-occurring in a mammal, is derived from a precursor protein or a mature protein and the second portion comprises a T helper cell epitope; or,

wherein the first portion comprises a T helper cell epitope and the second portion is from the free C-terminus of said naturally-occurring internal peptide cleavage product.

70. (Previously presented) The chimeric peptide according to claim 69, wherein said internal cleavage product is an amyloid β peptide, which when naturally-occurring, is derived from cleavage of β amyloid precursor protein (βAPP).

71. (Previously presented) The chimeric peptide according to claim 70, wherein said internal peptide cleavage product has an amino acid sequence selected from the group consisting of Aβ1-39, Aβ1-40, Aβ1-41, Aβ1-42, and Aβ1-43.

72. (Previously presented) The chimeric peptide according to claim 69, wherein the first portion is Aβ1-3, Aβ1-4, or Aβ1-5 from the free N-terminus of said internal peptide cleavage product.

73. (Previously presented) The chimeric peptide according to claim 69, wherein the first portion is Aβ35-40 or Aβ35-42 from the free C-terminus of said internal peptide cleavage product.

74. (Previously presented) The chimeric peptide according to claim 69, wherein said T helper cell epitope binds to multiple MHC molecules.

75. (Previously presented) The chimeric peptide according to claim 69, wherein said T helper cell epitope is derived from tetanus toxoid, diphtheria toxoid, hepatitis B surface antigen, Malaria CS, *E. coli* toxoid, or a toxoid from other pathogenic bacteria.

76. (Previously presented) The chimeric peptide according to claim 75, wherein said T helper cell epitope has an amino acid sequence selected from the group consisting of SEQ ID NO: 45, SEQ ID NO: 46, SEQ ID NO: 49, and SEQ ID NO: 50.

77. (Previously presented) An immunogenic composition, comprising an immunogenically effective amount of the chimeric peptide according to claim 69 and a pharmaceutically acceptable carrier, excipient, diluent, or adjuvant.

78. (Previously presented) The immunogenic composition according to claim 77, wherein said adjuvant is alum.

79. (New) A method for in vivo down-regulation of beta amyloid (Aβ) in a patient, including a human being, the method comprising effecting presentation to the patient's immune system of an immunogenically effective amount of at least one analog of Aβ that incorporates into the same molecule at least one B-cell epitope of Aβ and at least one foreign T-helper epitope (T_H epitope) so that immunization of the animal with the analog induces production of antibodies against the patient's endogenous Aβ, wherein the analog

a) is a polyamino acid that consists of at least one copy of a subsequence of Aβ, wherein the foreign T_H epitope is incorporated by means of amino acid addition and/or insertion and/or deletion and/or substitution, wherein the subsequence is selected

from the group consisting of residues 1-42, residues 1-40, residues 1-39, residues 1-28, residues 1-12, residues 1-5, residues 13-28, residues 17-28, residues 25-35, residues 35-40, and residues 35-42 of Aβ; and/or

b) is a polyamino acid that contains the foreign T_H epitopes and a disrupted Aβ sequence so that the analog does not include any subsequence of Aβ that binds productively to MHC class II molecules initiating a T-cell response; and/or

c) is a polyamino acid that comprises the foreign T_H epitope and Aβ derived amino acids, and comprises a conservative substitution.

80. (New) The method according to claim 79, wherein a substantial fraction of B-cell epitopes of Aβ are preserved in the analog and wherein at least one first moiety is introduced which effects targeting of the analog to an antigen presenting cell (APC) or a B-lymphocyte, and/or at least one second moiety is introduced which stimulates the immune system, and/or at least one third moiety is introduced which optimizes presentation of the analog to the immune system.

81. (New) The method according to claim 80, wherein the first and/or the second and/or the third moiety is/are attached as side groups by covalent or non-covalent binding to suitable chemical groups in the Aβ sequence.

82. (New) The method according to claim 79, wherein the analog comprises a fusion polypeptide.

83. (New) The method according to claim 79, wherein analog comprises conservative amino acid substitutions of the Aβ sequence.

84. (New) The method according to claim 79, wherein the analog includes duplication of at least one B-cell epitope of Aβ and/or introduction of a hapten.

85. (New) The method according to claim 79, wherein the foreign T-cell epitope is immunodominant in the patient.

86. (New) The method according to claim 79, wherein the foreign T-cell epitope is promiscuous, such as a foreign T-cell epitope which is selected from a natural promiscuous T-cell epitope and an artificial MHC-II binding peptide sequence.

87. (New) The method according to claim 86, wherein the natural T-cell epitope is selected from a Tetanus toxoid epitope such as P2 or P30, a diphtheria toxoid epitope, or an influenza virus hemagluttinin epitope.

88. (New) The method according to claim 79, wherein the analog comprises B-cell epitopes which are not exposed to the extracellular phase when present in a cell-bound form of a precursor polypeptide of Aβ.

89. (New) The method according to claim 79, wherein the analog lacks at least one B-cell epitope which is exposed to the extracellular phase when present in a cell-bound form of a precursor polypeptide of Aβ.

90. (New) The method according to claim 79, wherein the analog comprises at most 9 consecutive amino acids of Aβ.

91. (New) The method according to claim 90, wherein the analog comprises at least one subsequence of Aβ so that each such at least one subsequence of Aβ independently consists of amino acid stretches selected from the group consisting of 9 consecutive amino acids of Aβ, 8 consecutive amino acids of Aβ, 7 consecutive amino acids of Aβ, 6 consecutive amino acids of Aβ, 5 consecutive amino acids of Aβ, and 3 consecutive amino acids of Aβ.

92. (New) The method according to claim 90, wherein the consecutive amino acids begin at an amino acid residue selected from the group consisting of residue 1, 2, 3, 6, 13, 17, 25, 25, 33 and 35.

93. (New) The method according to claim 79, wherein presentation to the immune system is effected by having at least two copies of an Aβ derived fragment or the analog covalently or non-covalently linked to a carrier molecule capable of effecting presentation of multiple copies of antigenic determinants.

94. (New) The method according to claim 79, wherein the analog has been formulated with an adjuvant that enhances production of antibodies against the patient's endogenous Aβ.

95. (New) The method according to claim 79, wherein an effective amount of the analog is administered to the patient via a route selected from a parenteral route, and an intramuscular route; a peritoneal route; an oral route; an anal route; and an intracranial route.

96. (New) The method according to claim 95, wherein the parenteral route is intracutaneous or subcutaneous administration.

97. (New) The method according to claim 95, wherein the effective amount is between 0.5 μg and 2,000 μg of the analog.

98. (New) The method according to claim 79, wherein presentation of the analog to the immune system is effected by introducing one or more nucleic acids encoding the analog into the patent's cells and thereby obtaining in vivo expression by the cells of the one or more nucleic acids introduced.

99. (New) The method according to claim 98, wherein the one or more nucleic acids introduced are selected from naked DNA, DNA formulated with charged or uncharged lipids, DNA formulated in liposomes, DNA included in a viral vector, DNA formulated with a transfection-facilitating protein or polypeptide, DNA formulated with a targeting protein or polypeptide, DNA formulated with Calcium precipitating agents, DNA coupled to an inert carrier molecule, DNA encapsulated in chitin or chitosan, and DNA formulated with an adjuvant.

100. (New) The method according to claim 95, wherein an effective amount of the analog is administered at a frequency of at least one administration or introduction per year.

101. (New) A method for treating and/or preventing and/or ameliorating Alzheimer's disease or other diseases and conditions characterized by amyloid deposits, the method comprising down-regulating Aβ according to the method of any one of claims 79-100 to such an extent that the total amount of amyloid is decreased or that the rate of amyloid formation is reduced with clinical significance.

102. (New) An analog of Aβ which is derived from an patient Aβ wherein is introduced a modification which has as a result that immunization of the patient with the analog induces production of antibodies against the patient's endogenous Aβ, and wherein the analog is as defined claim 79.

103. (New) An immunogenic composition comprising an immunogenically effective amount of an analog according to claim 102, the composition further comprising a pharmaceutically and immunologically acceptable carrier and/or vehicle and optionally an adjuvant.

104. (New) A nucleic acid fragment which encodes an analog according to claim 102.

105. (New) A vector carrying the nucleic acid fragment according to claim 104, such as a vector that is capable of autonomous replication.

106. (New) The vector according to claim 105 which is selected from the group consisting of a plasmid, a phage and a virus.

107. (New) The vector according to claim 105, comprising, in the 5'→3' direction and in operable linkage, a promoter for driving expression of the nucleic acid fragment, optionally a nucleic acid sequence encoding a leader peptide enabling secretion of or integration

into the membrane of the polypeptide fragment, the nucleic acid fragment, and optionally a terminator.

108. (New) The vector according to claim 105 wherein, when introduced into a host cell, the vector is capable or incapable of being integrated in the host cell genome.

109. (New) The vector according to claim 107, wherein the promoter drives expression in a eukaryotic cell and/or in a prokaryotic cell.

110. (New) A transformed cell carrying the vector of claim 105.

111. (New) The transformed cell of claim 110, wherein the cell is capable of replicating the nucleic acid fragment.

112. (New) The transformed cell according to claim 110, wherein the cell is a microorganism selected from a bacterium, a yeast, or a cell derived from a multicellular organism selected from an insect cell, and a mammalian cell.

113. (New) The transformed cell according to claim 110, wherein the cell expresses the nucleic acid fragment.

114. (New) The transformed cell of claim 113, wherein the cell secretes or carries on its surface the analog.

115. (New) The method according to claim 79, wherein presentation to the immune system is effected by administering a virus which is carrying a nucleic acid fragment which encodes and expresses the analog.

116. (New) A composition for inducing production of antibodies against amyloid, the composition comprising a nucleic acid fragment according to claim 104 or a vector according to claim 105, and a pharmaceutically and immunologically acceptable carrier and/or vehicle and/or adjuvant.

117. (New) A stable cell line which carries the vector according to claim 105 and which expresses the nucleic acid fragment, and which optionally secretes or carries the analog on its surface.

118. (New) The method of claim 79, wherein the patient is a human.